Chlamydia Psittaci Infections in Man

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Introduction

The World Health Organisation lists some 200 zoonotic diseases but only 2, psittacosis and salmoneliosis are contracted directly from birds.[1] *Chlamydia psittaci is* one of three species of the genus Chlamydia. The other two are *C. trachomatis* and *C. pneumoniae* (formerly considered the TWAR agent and a strain of C. *psittaci).* [2]

Chlamydia are obligate intracellular bacteria with a cell wall similar to gram negative bacteria.[1] Elemental bodies (EB) about 350nm in diameter are phagocytosed by host cells but are not digested by lysosomes. The EB reorganises into a larger, metabolically active reticulate body (RB) about 1000nm in diameter which divides by binary fission. As the host cell becomes debilitated the RBs metamorphose into infectious RBs and are released upon the death of the host cell.

C. psittaci is a well known zoonotic agent infecting a wide variety of avian and mammalian hosts. It is most commonly associated with psittacines (parrots), other seed eating birds and poultry (particularly turkeys). Other animal reservoirs include cattle, sheep, goats, cats and koalas. The disease in humans is commonly referred to as psittacosis.

Seventy four cases of psittacosis were reported in Australia in 1993. [3]Most human cases had direct contact with birds. Strains of *C. psittaci* from psittacine birds and turkeys are most virulent for humans.[4] Carrier state prevalence may increase to 100% of bird populations stressed by overcrowding, breeding, transport, or intercurrent disease.

Over 130 avian species have been documented as hosts of *C. psittaci.*[5] These include parrots, finches, pigeons, pheasants, egrets, seagulls and poultry. Infected birds may be asymptomatic or clinically ill. Sick birds usually show closed eyes, ruffled feathers, depression and diarrhoea. They excrete the largest numbers of infectious organisms during periods of clinical illness. Both droppings and discharges from beaks and eyes are infective. The major method of spread is by aerosols of dust from the bird or its cage.

Humans at most risk of contracting psittacosis from work or leisure related exposures to animals include veterinarians, bird owners, pet shop employees, turkey farmers and turkey abattoir workers.[2]

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Clinical Signs

The incubation period for psittacosis is usually 5-15 days.[2] Onset may be insidious or abrupt and the clinical manifestations tend to be non-specific. Severity ranges from inapparent disease to a fatal systemic illness with prominent respiratory signs.

In Australia Yung and Grayson[6] reviewed 135 cases of serologically confirmed psittacosis admitted to Fairfield hospital, Melbourne between 1972 and 1986. They found psittacosis to be characterised by an abrupt onset of fever, rigors, sweats and prominent headache. Frequently a mild dry cough appeared late in the clinical course of the disease. Over 90% had crepitus of the lungs. Less frequently observed signs include photophobia (16%) hepatomegaly (10%) and splenomegaly (8%). A confused mental state (12%) and stiff neck (9%) were also noted.

Case fatality rate in the pre-antibiotic era was around 35%-40%.[7] However, with appropriate ant~iotic treatment it is around 1%.[1] Reinfection, chronicity and recurrence is not usually acknowledged, although the published literature contains some reports.[9,10]

Differential Diagnosis

The list of differential diagnoses is extensive and depends on presentation.[2] Fever, headache and respiratory signs are suggestive of atypical pneumonia which can also be caused by viral pneumonia, Q fever legionellosis and mycoplasmosis. Other presenting syndromes may resemble a non-specific viral illness, a mononucleosis-like syndrome and a typhoid-like syndrome. Illness caused by C. *pneumoniae* is a mild upper or lower respiratory disease widespread in most communities and unlikely to cause the severe type of illness reviewed here. Recent studies suggest it is newly recognised rather than a new disease. Transmission is human to human and no avian reservoirs are involved.[2]

Pathology

The lung is the major target organ in man. A sequence of pulmonary congestion, oedema and consolidation often in a lobular pattern is seen. Any lobe or lobes may be affected. There are alveolar and interstitial exudates of mononuclear cells, hyperplasia, proliferation and desquamation of alveolar lining cells. Intracytoplasmic inclusions maybe seen in alveolar lining cells. Lymph nodes may become oedematous. The brain may show congestion, oedema and a diffuse non-suppurative meningitis. The heart may show monocytic infiltration, sub-endocardial haemorrhage, oedema and fatty degeneration. The liver may demonstrate a diffuse or granulomatous hepatitis. Clinical pathological features may include marked elevation of the erythrocyte sedimentation rate, toxic granulation or left shift of neutrophils. Approximately 50% of cases have mild elevations of serum levels of hepatic enzymes.

Treatment

Tetracyclines are the treatment of choice both in humans and birds. In man, tetracycline hydrochloride 500mg qid or doxycycline 100mg bid orally for at least 2 weeks is recommended. Erythomycin is the alternative treatment (particularly where legionellosis cannot be excluded) but may be less efficacious in severe cases.[2] Most patients respond rapidly (within 24 hours) to appropriate therapy. However failure to respond to initial penicillin, amoxycillin or co-trimoxazole therapy was a commonly reported finding at the time of hospital admission.[6]

Diagnosis

A four-fold rise in complement fixing (CF) ant~odies is the established method of diagnosis. The centres for Disease Control and Prevention (CDC) in the USA regard a confirmed case as one with a positive culture or a clinical illness compat~le with psittacosis and a four-fold or greater rise in CF titre to at least 1:32. A presumptive case is a compatible illness with a CF titre of at lease 1:32 in a single specimen.

The CF test is genus specific and does not distinguish between *C. psittaci, C trachomatis* and C. pneumoniae.

IgM antibody may be detected. There are false positives and false negatives in both tests. Serological testing is imperfect and better tests are required.[2]

Discussion

Anecdotal evidence suggests that psittacosis is common and under-reported in veterinarians in Australia -particularly those in regular contact with caged and wild birds. Serological diagnosis can be erratic, particularly in the face of early antibiotic therapy. [1] The sensitivity of the CF test often varies between laboratories. The antigen used is critical and often guinea pig complement contains antibodies to chlamydia.[12]

Recrudescence and reinfection are thought not to be common. For example, there were no repeated infections in the 135 cases viewed in Australia by Yung and Grayson.[6] However, there is anecdotal evidence amongst veterinarians in NSW and limited evidence from the literature[8-10] to support both. Recrudescence and reinfection appears to be associated with delays in implementing ant~iotic therapy and withdrawing therapy too quickly. The presence of complement fixing ant~odies does not seem to confer protection against reinfection. [1,13] A human carrier state has been described [14] where a laboratory worked with a chronic pneumonic illness had C. *psittaci* isolated from his sputum several times over an 8 year period between 1938 and 1946. His CF titre varied from 1:64 to 1:256. Presumably effective tetracycline therapy would reduce or eliminate a carrier state - however, there is no direct evidence for this as culture is rarely attempted.

Conclusion

Psittacosis is a common disease in people who have regular contact with birds. It often presents as a distinct illness characterised by high fever, headache, rigor and sweats. Pneumonic involvement is common. The illness responds rapidly to tetracycline therapy which can be used as a diagnostic aid. Serological confirmation is possible in most but not all cases. Anecdotal evidence amongst veterinarians in NSW suggests recrudescences and reinfections are common.

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